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Research paper

The role of outcome expectancy in therapeutic change across psychotherapy versus pharmacotherapy for depression

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ABSTRACT

Background: Patient outcome expectancy - the belief that treatment will lead to an improvement in symptoms – is linked to favourable therapeutic outcomes in major depressive disorder (MDD). The present study extends this literature by investigating the temporal dynamics of expectancy, and by exploring whether expectancy *during* treatment is linked to differential outcomes across treatment modalities, for both optimistic versus pessimistic expectancy.

Methods: A total of 104 patients with MDD were randomized to receive either cognitive behavioral therapy (CBT) or pharmacotherapy for 16 weeks. Outcome expectancy was measured throughout treatment using the Depression Change Expectancy Scale (DCES). Depression severity was measured using both the Hamilton Depression Rating Scale and Beck Depression Inventory-II.

Results: Latent growth curve models supported improvement in expectancy across both treatments. Cross-lagged panel models revealed that both higher optimistic and lower pessimistic expectancy at mid-treatment predicted greater treatment response in pharmacotherapy. For CBT, the associative patterns between expectancy and depression differed as a function of expectancy type; higher optimistic expectancy at pre-treatment and lower pessimistic expectancy at mid-treatment predicted greater treatment response.

Limitations: The sample size limited statistical power and the complexity of models that could be explored.

Conclusions: Results suggest that outcome expectancy improved during treatment for depression. Whether outcome expectancy represents a specific mechanism for the reduction of depression warrants further investigation.

1. Introduction

Depression remains the most common mental disorder, with nearly 298 million cases of major depressive disorder (MDD) reported worldwide in 2010 (Ferrari et al., 2013). Although empirically supported treatments for MDD exist, including pharmacological and psychological interventions (Lam et al., 2009; Parikh et al., 2016), it is well recognized that a sizable proportion of patients do not respond to these treatments (Hofmann et al., 2012; Trivedi et al., 2006). Such findings highlight the need for a greater understanding of factors that predict response to treatment in MDD and the mechanisms by which treatment results in therapeutic change.

Patient expectancies of treatment outcome – that is, prognostic beliefs of whether treatment will lead to a change in health status - have been theoretically and empirically linked to both process and outcome in psychotherapy (Constantino et al., 2011; Glass et al., 2001; Greenberg et al., 2006) and pharmacotherapy (Rutherford et al., 2010). Indeed, theoretical models of psychotherapy including Frank's (1961) theory of remoralization and the Snyder et al. (2000) theory of hope posit a central role of patient expectancies in symptom improvement (Constantino et al., 2012; Swift and Derthick, 2013; Westra et al., 2007). Similarly, expectancy theory posits that conscious expectancies of improvement mediate the placebo response seen in pharmacological treatments (Rutherford and Roose, 2013; Stewart-Williams and Podd,

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2004). As overall treatment efficacy has been conceptualized as the sum of the placebo effect and the effects specific to the active pharmacotherapy, more optimistic expectancy may contribute to stronger treatment response with active medication (Wampold et al., 2005). This link between outcome expectancy and treatment response has been demonstrated consistently in individuals with a diagnosis of depression (Schulte, 2008; Webb et al., 2013).

The majority of the empirical support for the influence of patient outcome expectancies has been circumscribed to psychotherapy. In a comprehensive meta-analysis of 46 independent studies, outcome expectancy at the beginning of psychotherapy was found to have a small, significant positive association with therapeutic outcomes, such as post-treatment symptom severity (Cohen's $d = 0.24$; Constantino et al., 2011). More recent research supports the dynamic nature of this construct within psychotherapy treatment in anxious (Brown et al., 2014; Newman and Fisher, 2010) and obsessive-compulsive samples (Vorstenbosch and Laposa, 2015). Change in this construct has also been demonstrated for treatment of mood disorders. In individuals with recurrent major depression with a seasonal pattern, outcome expectancy increased steeply over a 6-week course of group CBT for seasonal affective disorder (SAD) (Meyerhoff and Rohan, 2016). Finally, increased outcome expectancy was recently demonstrated in patients with depression who received a 14-week course of individual CBT (Vîslă et al., 2018).

This growing literature of dynamic changes in patient expectancy highlights the importance of evaluating outcome expectancy not only at pre-treatment, but also during treatment course. The demonstration of an association between expectancy *during* treatment and subsequent symptom improvement may substantiate the mechanistic role of expectancy. Indeed, change in expectancy during psychotherapy has predicted symptom severity and functioning level at post-treatment in several studies (Brown et al., 2014; Newman and Fisher, 2010; Vorstenbosch and Laposa, 2015), whereas point estimates of outcome expectancy during treatment were linked to clinical outcomes in others (Meyerhoff and Rohan, 2016; Visla et al., 2016).

Nevertheless, important questions remain in the literature. First, the causal significance of this construct across different treatment modalities is presently unclear (Meyerhoff and Rohan, 2016). Comparing the impact of outcome expectancy across different classes of treatment can help to clarify whether expectancy of change is a putative common mechanism for improvement, or whether it is a possible mechanism specific to treatment change. To this end, exploring the role of outcome expectancy across cognitive behavioral therapy (CBT) and pharmacotherapy, two treatments for depression for which there are comparable outcomes and among the strongest evidence base (Cuijpers et al., 2013), is warranted. Like CBT, there is considerable evidence for the predictive power of pre-treatment expectancy on therapeutic response in pharmacotherapy (Meyer et al., 2002; Rutherford et al., 2017, 2010; Sotsky et al., 1991), although expectancy *during* pharmacotherapy treatment has received limited investigation.

Second, the measurement of patient outcome expectancy is consistently recognized as a limitation in the literature to date. As reviewed by Rutherford et al. (2010), the majority of self-report scales developed for the assessment of outcome expectancy were purpose-built for a specific investigation, with limited psychometric validation beyond those investigations. A further important consideration is that expectancies can be directed to the likelihood of positive outcomes (i.e., optimistic expectancy) as well as negative outcomes (i.e., pessimistic expectancy; Schulte, 2008), although existing instruments largely assess only the former. The existence of optimistic and pessimistic expectancies is highly relevant to MDD in particular; both optimistic and pessimistic cognitions are seen in this disorder in the form of certainty in the absence of positive future events (i.e., low optimistic expectancy) and the presence of negative future events (i.e., high pessimistic expectancy; Miranda et al., 2008), each proposed to stem from distinct underlying affective-motivational systems (MacLeod, 1996). In the context of depression, there is thus a particular need to ascertain both optimistic and pessimistic expectations of outcomes (Dozois and Westra, 2005; Eddington et al., 2014; Schulte, 2008).

The current study adds to the growing expectancy literature by first investigating the temporal dynamics of outcome expectancy during the course of different treatments for MDD. Specifically, growth is compared across CBT and pharmacotherapy as these treatments represent empirically supported interventions for MDD with theoretically distinct mechanisms of action. Second, we investigate whether outcome expectancy both *before* and *during treatment* is predictive of depression outcome in each treatment. Finally, this study improves upon the current literature by measuring outcome expectancy using a more refined scale (the Depression Change Expectancy Scale (DCES) (Eddington et al., 2014)) designed to evaluate both pessimistic and optimistic expectancies specific to depression.

Theoretically, CBT improves depression by targeting cognitions *via* both cognitive and behavioral strategies. As reviewed by Lorenzo-Luaces et al. (2015), there is some evidence that depressive cognitions undergo the greatest improvements as a result of procedures specifically aimed at cognitive change such as those used in CBT. Although cognitive change can also occur in pharmacotherapy (see Garratt et al., 2007, for review), such change likely stems from other mechanisms (see Lorenzo-Luaces et al., 2015); the mechanisms of action of antidepressants have largely implicated neurobiological targets (Rot et al., 2009). As such, cognitive changes in pharmacotherapy may represent "downstream effects", or may be a reflection of symptom change itself (DeRubeis et al., 1990). Based on the conceptualization of outcome expectancy as a cognitive variable, we hypothesize that this variable will undergo greater change over treatment in the CBT arm relative to pharmacotherapy. We also hypothesize that the association between outcome expectancy during treatment and symptom improvement will be stronger in the CBT arm relative to pharmacotherapy treatment. This hypothesis is first supported by the centrality of positive expectations to theories of depression improvement in psychotherapy (Frank, 1961). Moreover, there is some empirical support that cognitive change is causally linked to symptom change within the context of therapies utilizing cognitive-specific procedures (i.e., CBT) and less so in therapies using non-cognitive strategies (DeRubeis et al., 1990; Evans et al., 2013; Lorenzo-Luaces et al., 2015). Due to limited empirical evidence, no specific hypotheses were made with respect to the differing role of optimistic versus pessimistic expectancy.

2. Methods

2.1. Participants

Participants were 104 outpatients diagnosed with MDD who participated in a randomized trial of CBT versus pharmacotherapy (Quilty et al., 2014). Participants met the following inclusion criteria: (i) having a primary diagnosis of MDD as determined using the *Structured Clinical Interview for DSM-IV Patient Version* (SCID-I/P; (First et al., 1995)); (ii) being between 18 and 65 years of age; (iii) fluency in English; and (iv) capacity to provide consent. Moreover, participants: (i) did not have a diagnosis of bipolar disorder, psychotic disorder, substance dependence or organic brain syndrome; (ii) were not receiving current treatment with antidepressant medications, and (iii) did not receive electroconvulsive therapy in the 6-month period prior to enrollment.

The randomized trial of CBT versus pharmacotherapy (Quilty et al., 2014) was undertaken to explore the cognitive mediation hypothesis of treatment for depression, specifically the mediational role of cognitive structure and processing. Quilty et al. reported that both indices of cognition improved similarly across CBT and pharmacotherapy but did not evaluate measures of cognitive content, including outcome expectancy.

2.2. Procedures

Participants were recruited using a range of advertisement approaches. The 104 participants provided written consent to participate and were randomized to receive either CBT (n = 54), or antidepressant medication (n = 50). Stratified randomization was used to match participants on gender and depression recurrence across both treatments. There were 5 dropouts in the CBT group and 7 dropouts in the medication group, resulting in 49 CBT treatment completers and 43 medication treatment completers (final N = 92). Treatment completers had completed at least 8 sessions of CBT or weeks of medication.

Four psychiatrists provided treatment in the antidepressant arm, guided by the Canadian Network for Mood and Anxiety Treatment (CANMAT) guidelines (Lam et al., 2009). Specifically, the pharmacotherapy protocol included eight bi-weekly psychiatric visits approximately 30 min in duration, and medication switches as per CANMAT guidelines. Participants in this arm received 13.5 weeks of medication on average (*range*: 0–16 weeks). Medications included bupropion (n = 12), sertraline (n = 10), venlafaxine (n = 6), citalopram (n = 6), escitalopram (n = 5), fluoxetine (n = 5), mirtazapine (n = 4), and duloxetine (n = 2); six participants were on more than one antidepressant. Moreover, they attended 6.5 psychiatric visits on average (*range*: 0–9 visits).

Nine therapists provided CBT, guided by the protocol outlined by Beck and colleagues (Beck et al., 1979). The CBT protocol involved sixteen weekly visits lasting approximately one hour in duration. Participants in the CBT group completed 14.3 sessions on average (*range*: 1 – 16 visits).

Intraclass correlation coefficients (ICCs) were computed to estimate the proportion of variance in the variables of interest (outcome expectancy and depression) explained by assignment to specific therapists/psychiatrists. The ICC values were modest (0.002 to 0.08), not supporting the need for multi-level models incorporating the effect of therapists.

2.3. Measures

During treatment, assessments were conducted at four time points: at pre-treatment (week 0), week 4, week 8 and post-treatment (week 16; Quilty et al., 2014). Outcome expectancy was measured using the DCES (Eddington et al., 2014). The DCES is a recently developed self-report questionnaire developed for the measurement of outcome/change expectancy in depression, modified from an expectancy scale developed for anxiety disorders (Dozois and Westra, 2005). The DCES assesses both broad as well as treatment-focused expectations for change in depression symptoms, using a Likert-type scale ranging from 1 (strongly disagree) to 5 (strongly agree). The DCES comprises the DCES-P and the DCES-O subscales, which include 11 pessimistically worded items and 9 optimistically worded items, respectively. The DCES-P items were reverse scored. In a psychometric evaluation using a clinical sample, the DCES demonstrated good internal consistency (coefficient α = 0.75 and 0.82) and expected convergent, divergent, and predictive validity with respect to short-term improvement in depression symptoms (Eddington et al., 2014).

Depression was measured using the self-reported Beck Depression Inventory-II (BDI-II; Beck et al., 1996) and the semi-structured, clinician rated Hamilton Depression Rating Scale (HAMD; Hamilton, 1960). The reliability of these measures within this sample is reported elsewhere (Quilty et al., 2014).

2.4. Data analyses

Descriptive statistics were tested using IBM SPSS Statistics Version 24.0, and analyses were conducted using Mplus Version 8.0. Model parameters were estimated using robust maximum likelihood estimation, which is robust to non-normality (Muthén and Muthén, 2017). Model fit was ascertained using the following fit indices: chi-square goodness of fit, standardized root mean square residual (SRMR), root mean-square error of approximation (RMSEA), and Bentler's Comparative Fit Index (CFI). Based on the recommendations of Hu and Bentler (1999), good model fit was defined as an RMSEA value of <0.05, SRMR value of <0.08 and a CFI value of >0.95. RMSEA values of <0.08 were considered indications of adequate model fit and RMSEA values of >0.10 were considered poor fit (Hu and Bentler, 1999; Kline, 2011). For all models, modification indices were examined upon indication of possible misspecification. Only theoretically tenable modifications were considered (Kline, 2011).

Change in outcome expectancy was investigated using latent growth curve modeling (LGCM) – a growth model approach to estimate between-participant differences in the latent trajectory of a variable (Curran et al., 2010). Two latent factors representing the intercept and slope were estimated, along with the covariance between the two factors. The mean of the intercept and slope represent fixed effects, indicating the average baseline value and average change over time, respectively. Variability in these factors between participants – i.e. random effects - are represented with the factor variances (Newsom, 2015). As demonstrated by Preacher et al. (2008), model fitting began with a random intercept model. A fixed slope parameter was then introduced, and then subsequently freed, allowing growth parameters to covary; model fit indices were explored at each step of model building (Preacher et al., 2008). Visual inspection of the pattern of change for the optimistic and pessimistic subscales of the DCES in the two treatment groups suggested non-linear growth only in the medication group. As such, both linear (slope loadings = 0, 1, 2, 4) and freely estimated (slope loadings = 0, *, *, 4) unconditional models were compared for each subscale in the two treatment groups separately.[1]

The relation between outcome expectancy and depression throughout treatment was investigated using cross-lagged panel models. Cross-lagged models allow for approximation of the causal relation between variables longitudinally, by accounting for both the stability within each variable and contemporaneous associations between variables. Thus, the potential covariation and confounding of expectancies and response (e.g., patients who are improving to a greater degree may endorse more positive expectancies) are taken into account statistically, and not reflected within cross-lagged associations. As equidistant time intervals are required to ensure unbiased interpretation of results (Kuiper and Ryan, 2018), only week 0, week 8 and week 16 were included. A multi-group approach was taken to model the panels separately in the medication and CBT group, allowing for an exploration of whether the cross-lagged associations differed as a function of treatment group. To limit the number of tests performed, the Wald Test of Parameter Constraints (Muthén and Muthén, 2017) was used to test the equality of the regression parameters in only the cross-lagged paths from expectancy to depression in the medication versus CBT treatment groups.

The following cross-lagged panel models were developed to assess the causal relation between outcome expectancy and depression over time: pessimistic outcome expectancy modeled with the BDI-II (Model 1) and the HAMD (Model 2), and optimistic outcome expectancy modeled with the BDI-II (Model 3) and the HAMD (Model 4). Model-building began with an initial structure applied to all four models, which included all autoregressive and cross-lagged panel effects with only a time lag of 1 (i.e., depression at time t on depression at time $t + 1$ and expectancy at $t + 1$).

[1] Consistent with the observed trends, the freely estimated slope demonstrated more favorable fit over the linear slope on all indices for the medication group. For the CBT group, the fit of the two conditional models (linear slope versus freely estimated slope) was comparable. Importantly, the freely estimated slopes approximated linear change in the CBT group (i.e. estimated slope loadings = 0, 1.2, 2.4, 4), supporting the observed pattern of change.

Table 1
Sample descriptive statistics for depression change expectancy scale measure.

Variables	Medication Means	*SD*	CBT Means	*SD*
DCES-O week 0	29.00	5.83	30.52	5.73
DCES-O week 4	31.74	5.64	32.40	4.82
DCES-O week 8	32.85	6.70	34.16	4.60
DCES-O week 16	33.33	7.44	36.54	6.09*
DCES-P week 0	35.93	8.23	34.37	8.68
DCES-P week 4	39.33	8.58	37.50	7.37
DCES-P week 8	39.87	10.62	41.36	7.93
DCES-P week 16	42.38	10.51	46.21	6.64

Note: DCES-O, optimistic outcome expectancy subscale of DCES; DCES-P, pessimistic outcome expectancy subscale of DCES. DCES-P items were reverse scored. *Significant group difference, $p < .05$.

3. Results

3.1. Change in outcome expectancy

Neither outcome expectancy (DCES-P and DCES-O) nor depression (BDI-II and HAMD) differed between treatment groups at week 0 (*ps* > 0.05; Table 1). We could not assume measurement invariance across our treatment groups owing to the differing shapes of the trajectories (and therefore, non-invariant factor loadings; Kim and Willson, 2014; Newsom, 2015; Preacher et al., 2008). As such, the latent growth curve model (LGCM) analyses were estimated in the CBT and medication groups separately. Model fits of all final models are reported in Table 2. Unstandardized parameter estimates from all LGCMs are reported in Table 3.

3.1.1. Medication

The LGCM for pessimistic outcome expectancy including first a fixed slope, and then a random slope resulted in a poor fitting and an inadmissible model solution, respectively. This indicated potential structural misspecification requiring modification, with the model deemed uninterpretable. The LGCM for optimistic outcome expectancy including a random slope demonstrated the best fit and was used as the final model (Table 2). The mean slope indicated significant positive non-linear growth in optimistic outcome expectancy over treatment ($B = 1.05$, $p < .001$).

Table 2
Fit indices for latent growth curve and cross-lagged panel models.

Latent growth curve models (LGCMs) Treatment	Expectancy	χ^2 (df), *p* value	CFI	RMSEA	SRMR
Medication	DCES-P	–	–	–	–
	DCES-O	1.63 (3), $p = .65$	1.00	<0.01	0.08
CBT	DCES-P	3.97 (5), $p = .55$	1.00	<0.01	0.11
	DCES-O	4.82 (7), $p = .68$	1.00	<0.01	0.16

Multi-Group Cross-Lagged Panel Models (CLPMs) Model	Expectancy	Outcome	χ^2 (df), *p* value	CFI	RMSEA	SRMR
1.	DCES-P	BDI-II	2.46 (8), $p = .96$	1.00	<0.01	0.03
1.*			2.35 (4), $p = .67$	1.00	<0.01	0.03
2.		HAMD	7.46 (8), $p = .49$	1.00	<0.01	0.03
2.*			6.18 (4), $p = .19$	0.99	0.10	0.04
3.	DCES-O	BDI-II	14.84 (8), $p = .06$	0.96	0.13	0.06
3.*			2.36 (4), $p = .67$	1.00	<0.01	0.03
4.		HAMD	10.18 (8), $p = .25$	0.98	0.07	0.07
4.*			3.00 (4), $p = .56$	1.00	<0.01	0.03

Note: In the LGCMs, the model for DCES-P for the medication group is not presented due to notable misspecification; it was not interpreted.
*Final CLPMs, which include paths from week 0 to week 16.

3.1.2. Cognitive behavioral therapy

The LGCM for pessimistic outcome expectancy with a random slope demonstrated the best fit and was used as the final model (Table 2). The mean slope indicated significant positive linear growth ($B = 2.75$, $p < .001$). For optimistic outcome expectancy, introduction of a random slope factor resulted in an inadmissible model solution. As such, the final model included a fixed linear slope, which demonstrated acceptable model fit (Table 2). The mean slope indicated significant positive linear growth on average ($B = 1.47$, $p < .001$).

Taken together, results of the LGCMs supported non-linear growth in optimistic outcome expectancy within the medication group and linear growth in optimistic outcome expectancy and (reverse scored) pessimistic outcome expectancy in CBT. However, for most of the LGCMs, support for model fit was mixed, with the SRMR values outside the recommended cutoff (<0.08; see Table 2). As such, the presence of some model misfit is possible.

3.2. Links between outcome expectancy and depression

Fit indices for the initial lag 1 models are displayed in Table 2. Based on both extant literature substantiating the relation between pre-treatment expectancy and post-treatment outcome, and previous work with similar study aims (Meyerhoff and Rohan, 2016), cross-lagged paths between week 0 and week 16 for the four models were added.[2]

3.3. Pessimistic outcome expectancy and depression

All significant paths from Model 1 are shown in Fig. 1. Higher DCES-P score at mid-treatment predicted lower BDI-II score at the end of treatment in the CBT group ($\beta = -0.31$, $p = .02$). However, the Wald test was not significant ($\chi^2(1, n = 104) = 0.04$, $p = .85$), suggesting that the strength of the path did not significantly differ between treatment arms.

Using the HAMD to model depression (Model 2), higher DCES-P score at mid-treatment predicted lower HAMD score at the end of treatment in the medication group ($\beta = -0.43$, $p = .004$) as well as in the CBT group ($\beta = -0.32$, $p = .03$). Similar to the results of Model 1, the Wald test indicated that the estimated coefficients for this path did not significantly differ across the two arms ($\chi^2(1, n = 104) = 0.03$, $p = .87$). In both Models 1 and 2, none of the paths from depression to pessimistic outcome expectancy were significant in either of the two treatment groups (*ps* > 0.05).

3.4. Optimistic outcome expectancy and depression

All significant paths from Model 3 are shown in Fig. 2. Higher DCES-O score at pre-treatment predicted lower BDI-II score at mid-treatment in the medication group ($\beta = -0.30$, $p = .03$), but not the CBT group ($\beta = 0.03$, $p = .82$). However, the difference in this path across the two groups was not significant ($\chi^2(1, n = 104) = 3.13$, $p = .077$). Similarly, higher DCES-O scores at mid-treatment predicted lower BDI-II scores at the end of treatment in the medication group ($\beta = -0.30$, $p = .04$), but not the CBT group ($\beta = -0.13$, $p = .41$). Testing the difference in this path across treatment groups revealed no significant differences ($\chi^2(1, n = 104) = 0.623$, $p = .43$). Finally, higher DCES-O scores at pre-treatment predicted lower BDI-II scores at the end of treatment for the CBT group ($\beta = -0.35$, $p < .001$), but not the medication group ($\beta = 0.01$, $p = .89$). The Wald test in this particular path indicated significant differences between medication and CBT ($\chi^2(1, n = 104) = 6.07$, $p = .01$). In the medication group, none of the depression to outcome expectancy paths emerged as significant (*ps* > 0.05). However, in the CBT group, the path from BDI-II at week 8 to DCES-O at week 16 was significantly different from 0 ($\beta = 0.29$, $p = .03$).

[2] The addition of this path improved model fit for all models, with the exception of Model 2. To maintain consistency across models, the additional paths from week 0 to 16 were included in all four models.

Table 3
Parameter estimates for final latent growth curve models.

Model	Parameters	Medication		CBT	
DCES-P		**Mean (SE)**	**Variance (SE)**	**Mean (SE)**	**Variance (SE)**
	Intercept	_	_	34.54 (1.10)**	48.47(13.66)**
	Slope	_	_	2.75 (0.26)**	2.22 (1.27)
	Growth factor Correlation (r, 95% CI)	_		−0.48 [−0.75, −0.20]*	
DCES-O	Intercept	29.06 (0.81)**	20.96 (9.63)*	30.78 (0.59)**	9.13 (2.63)**
	Slope	1.05 (0.27)**	1.19 (0.62)	1.47 (0.21)**	_
	Growth factor Correlation (r, 95% CI)	−0.10 [−0.79, 0.59]		_	

Note: *$p < .05$, **$p < .001$. Unstandardized mean and variance estimates are reported. Standardized covariance (correlations) between growth factors are reported. The model for DCES-P growth in the medication group is not presented due to notable misspecification.

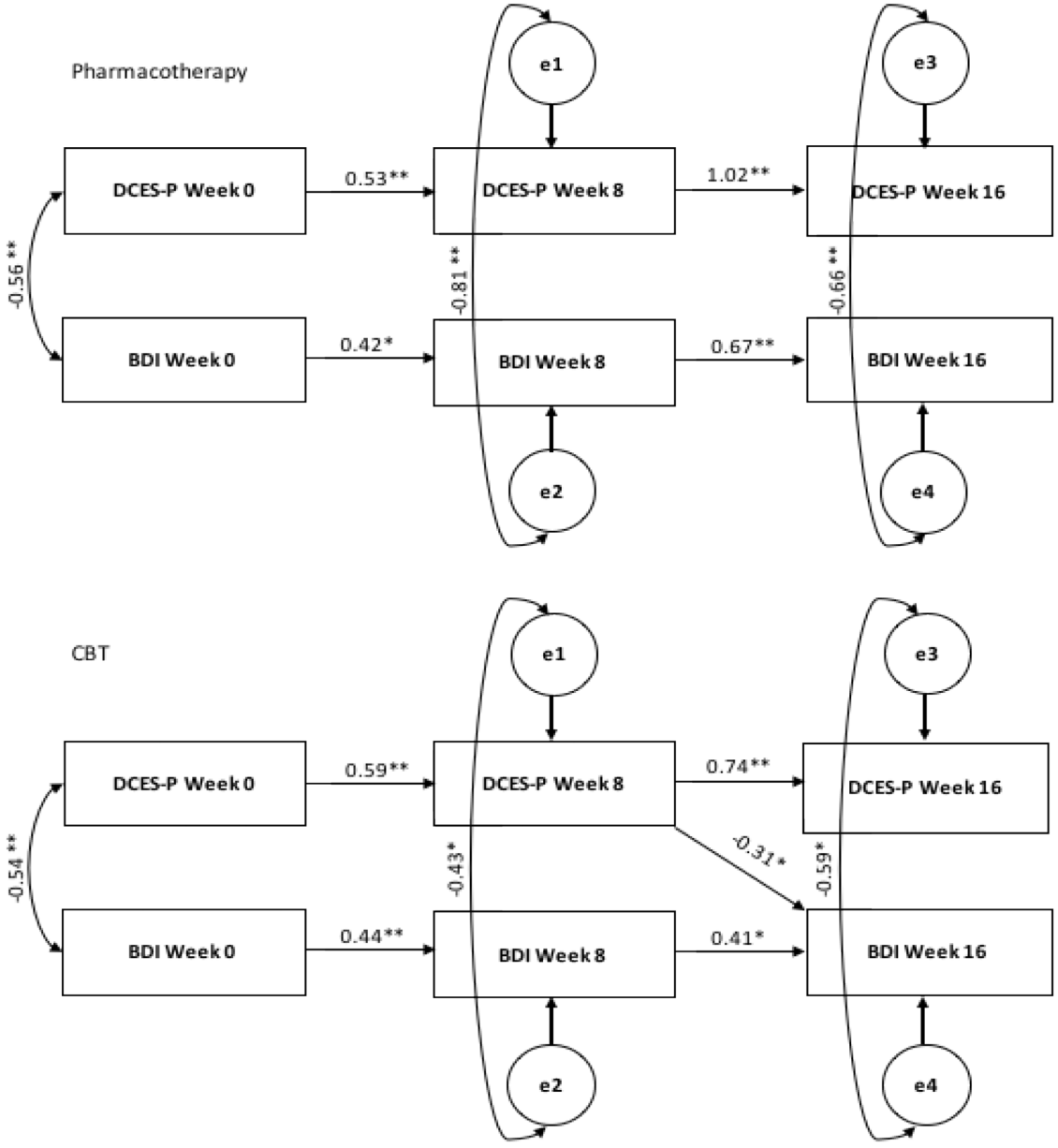

Fig. 1. Model 1 Cross-lagged panel model of DCES-P and BDI-II. Only significant paths are shown. e1-e4 = error residual terms. * = $p < .05$, ** = $p < .001$. Results of the Wald test did not support differences between treatment groups in the path from week 8 DCES-P and week 16 BDI-II ($\chi^2 = 0.04(1, n = 104)$, $p = .852$).

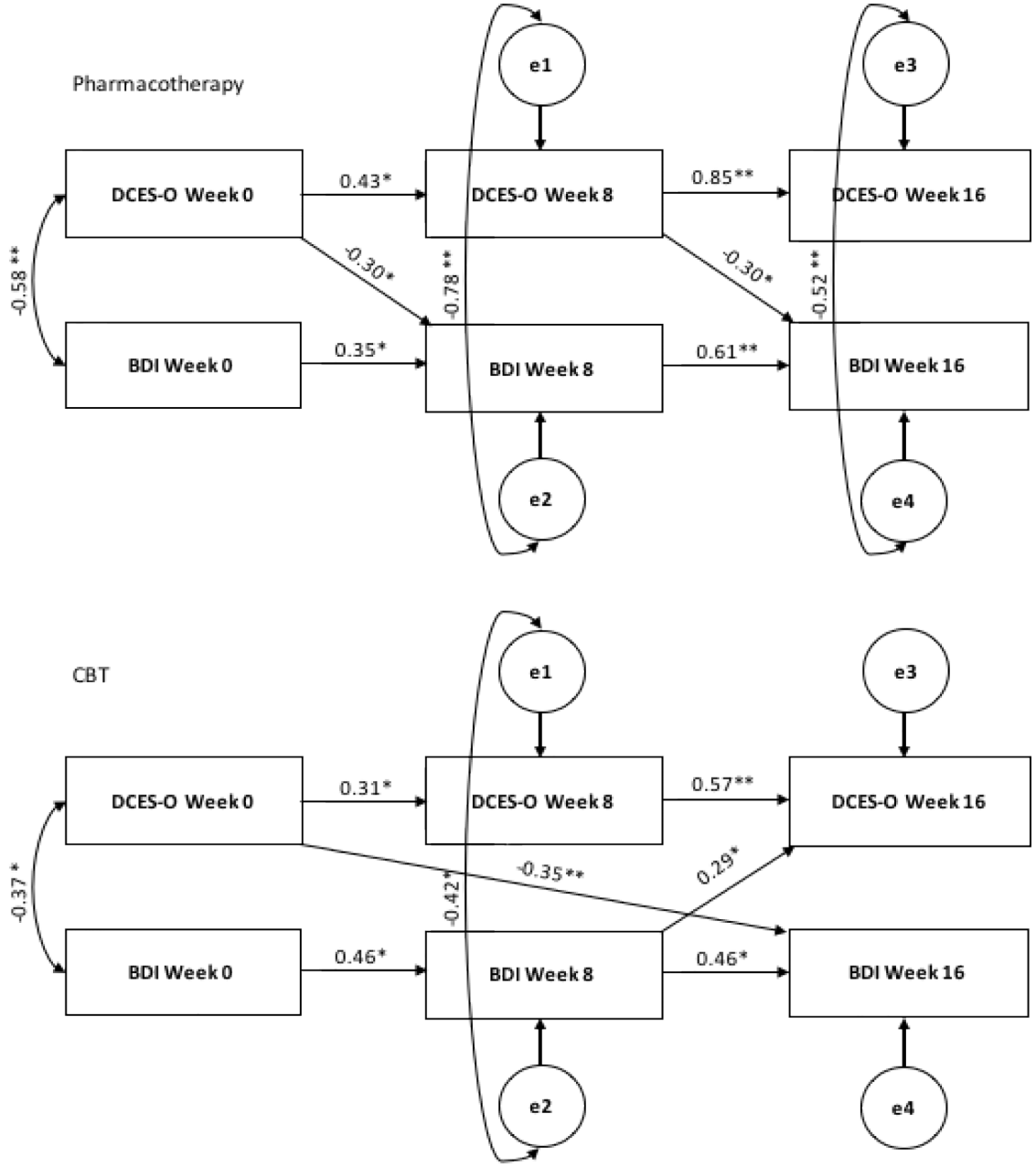

Fig. 2. Model 3 Cross-lagged panel model of DCES-O and BDI-II. Only significant paths are shown. e1-e4 = error residual terms. * = $p < .05$, ** = $p < .001$. Results of the Wald test did support differences between treatment groups in the path from week 0 DCES-O and week 16 BDI-II ($\chi^2 = 6.07$ (1, $n = 104$), $p = .014$).

Using the HAMD (Model 4), a significant difference between medication and CBT ($\chi^2(1, n = 104) = 5.61, p = .02$) was found in the path from mid-treatment DCES-O score to end of treatment HAMD score. That is, higher DCES-O at mid-treatment predicted lower end of treatment HAMD in the medication group ($\beta = -0.45$, $p < .001$), not the CBT group ($\beta = 0.030$, $p = .82$). Similar to Model 3, higher DCES-O at pre-treatment predicted lower HAMD scores at the end of treatment in the CBT arm only, although the Wald-test did not reach significance in Model 4 ($\chi^2(1, n = 104) = 1.46, p = .23$). Additionally, no group differences were seen in the paths between week 0 DCES-O and week 8 HAMD. Finally, similar to Model 3, there were no significant paths from depression to outcome expectancy in the medication group; however, HAMD at week 0 was negatively predictive of DCES-O at the end of treatment in the CBT group ($\beta = -0.28$, $p = .05$).

Taken together, similar patterns of the relation between optimistic outcome expectancy and depression were seen in the BDI-II (Model 3) and HAMD (Model 4) models. That is, for the medication group, higher DCES-O at mid-treatment predicted lower depression at the end of treatment. In the HAMD model, the results of the Wald test supported that this path was specific to treatment with medication over treatment with CBT. Moreover, both models demonstrated that higher pre-treatment optimistic expectancy predicted lower depression at the end of treatment. The results of the Wald test in the BDI-II model suggested that this effect of pre-treatment expectancy on post-treatment depression was specific to CBT over medication.[3]

[3] As age and sex demonstrated significant associations with depression and expectancy scores, these variables were included as independent covariates in the cross-lagged models. Incorporation of age as a covariate did not change study results. Incorporation of sex as a covariate in these models resulted in inadmissible solutions.

4. Discussion

The present study explored differences in the trajectory of outcome expectancy in pharmacotherapy versus CBT for depression. This randomized design enabled the evaluation of the role of outcome expectancy as a putative mechanism for depression reduction as these treatments progressed. The recently developed DCES permitted the examination of both optimistic expectancy and pessimistic expectancy in this context.

4.1. Change in expectancy over time

Results from latent growth model analyses supported significant positive, linear improvement in optimistic and pessimistic expectancy in CBT, reflecting consistent change throughout psychotherapy. In contrast, a clear significant quadratic trend in optimistic outcome expectancy was seen in pharmacotherapy, reflecting significant steep growth from week 0 to week 8, followed by a slight decrease by the end of the treatment. Increases in expectancy throughout the course of CBT has been established in previous investigations in patients with generalized anxiety disorder (Newman and Fisher, 2010), mixed anxiety disorders (Brown et al., 2014), seasonal affective disorder (Meyerhoff and Rohan, 2016), and most recently in MDD (Vîslă et al., 2018). To the best of our knowledge, this is the first investigation to examine the trend of patient expectancy during antidepressant treatment. Previous investigations of this cognitive construct in pharmacotherapeutic studies have largely been limited to exploring the influence of pre-treatment expectancy on end of treatment outcome (Krell et al., 2004; Meyer et al., 2002; Rutherford and Roose, 2013; Sotsky et al., 1991). Evidence for dynamic changes in expectancy as treatment progresses signals a potential mechanistic role of this construct to therapeutic improvement with pharmacotherapy.

Differing trajectories of growth across the two treatment modalities precluded direct comparisons of the growth parameters (slope and intercept) between groups, and may reflect either treatment-specific processes influencing change in this construct, or similar processes acting at different time points throughout the course of the two treatments. Although the extant literature on how expectancy changes over treatment for MDD is notably small, evidence is sufficient to suggest processes that are largely common across treatments.

In a recent study in which patients with MDD were randomized into either an open-label citalopram group (100% chance of active drug) or a placebo-controlled citalopram group (50% chance of active drug), revealing group assignment post-randomization enhanced patient outcome expectancy only in the open-label group (Rutherford et al., 2017). Both groups were informed of the effectiveness of citalopram for depression and only differed in the level of certainty in receiving this medication, providing some support for the importance of treatment rationale for early expectancy change in pharmacotherapy treatment. Moreover, there is growing evidence for the influence of therapeutic alliance on treatment outcomes in the placebo arm (i.e., robustness of placebo effect) (Rutherford and Roose, 2013; Zilcha-Mano et al., 2015). Insofar as patient expectations of improvement underscore placebo effects (Rutherford and Roose, 2013), building alliance may represent an important process to expectancy change in treatment with medication.

Relatedly, research investigating the influence of treatment rationale on patient expectations in CBT suggest that providing a rationale alone significantly increases expectancy in socially anxious analogue samples (Ahmed and Westra, 2009; Ametrano et al., 2017). As suggested by Newman and Fisher (2010), although the fulsome rationale is typically presented at the beginning of treatment, there are opportunities throughout CBT to reiterate elements of the rationale, particularly as new strategies are being introduced (Newman and Fisher, 2010) or homework is reviewed, facilitating consistent growth in expectancy. Second, CBT strategies during the latter half of treatment (i.e. cognitive restructuring and behavioral experiments) may directly influence this construct (Newman and Fisher, 2010). Finally, as in pharmacotherapy, there is some support for the effect of strong alliance on outcome expectations in CBT (Westra et al., 2011; Visla et al., 2016).

4.2. Outcome expectancy as a putative mechanism

Our hypothesis that outcome expectancy during treatment would demonstrate predictive associations with depression improvement in CBT, but not pharmacotherapy, was not supported by the results of the cross-lagged panel analyses. For the CBT group, greater pre-treatment optimistic expectancy (week 0) predicted lower post-treatment depression (week 16). In the pharmacotherapy group, higher optimistic expectancy at mid-treatment (week 8) was predictive of lower post-treatment depression. The relation between pessimistic expectancy and depression did not differ across treatment groups. Indeed, in both CBT and pharmacotherapy, lower pessimistic expectancy (seen as higher scores on the pessimistic subscale of the DCES) at mid-treatment predicted lower depression at the end of treatment.

Our results are consistent with previous work establishing a small effect between pre-treatment optimistic outcome expectancy and post-treatment symptom severity in psychotherapy (Constantino et al., 2011). This finding supports long-standing suggestions that the beginning of therapy with CBT is a crucial point in which to shape treatment outcome by strengthening optimistic expectancies for improvement (Constantino et al., 2012; Ilardi and Craighead, 1994). Previous work on the possible mechanistic processes linking pre-treatment expectancy and post-treatment outcome implicate alliance formation (Tsai et al., 2014; Visla et al., 2016; Webb et al., 2014), early homework compliance (Westra et al., 2007) and the use of CBT skills (Webb et al., 2013). Importantly, results from Visla et al. (2016) support the presence of chain associations *throughout* treatment by which baseline expectancy leads to early alliance formation which, in turn, influences early-treatment expectancy, leading to more favourable post-treatment outcomes. In some support of this idea, pre-treatment outcome expectancy (both optimistic and pessimistic) was significantly related to the number of sessions completed by participants in the CBT group (rs of 0.42 and 0.42, ps $= 0.001$). In contrast, pre-treatment expectancy was not related to the total number of weeks of medication received, or the total number of visits with the psychiatrist, in the pharmacotherapy group.

Our lack of association between during-treatment optimistic expectancy and depression outcomes in CBT is inconsistent with the results of a previous study, wherein a significant effect of higher week 3 expectancy on lower end of treatment depression was evidenced (Meyerhoff and Rohan, 2016). However, the aforementioned study differs from the present investigation in several respects, including their sample (seasonal depression only), treatment duration (six weeks in duration), and measurement of expectancy (which combined expectancy and credibility). These results may suggest that optimistic expectations during treatment are impactful only very early on where the processes of alliance formation and homework compliance gain the most traction. In line with these results, Visla et al. (2016) saw a robust negative association between week 3 expectancy and post-treatment depression at week 10.

Unlike optimistic expectancy, a negative association was seen in week 8 expectancy and post-treatment depression in CBT for pessimistic expectancy. Poor treatment outcome was seen in individuals who maintained higher pessimistic expectancies at the midpoint of treatment. This differential effect of optimistic versus pessimistic expectancies aligns with extant theories of goal attainment which postulate persistent effort stemming from positive expectancies, and disengagement/abandonment stemming from negative expectancies (Carver and Scheier, 2000; Meyer et al., 2002). Although optimistic and pessimistic outcome expectancy subscales were highly correlated in our study (rs $= 0.68$ to 0.76, ps < 0.001, across time points), the differential effects suggests that each subscale represents a unique

component of this construct, in line with the results of the original validation for this scale (Eddington et al., 2014). Future studies are needed to replicate the proposed differential roles of positive (optimistic) versus negative (pessimistic) expectancies in CBT, particularly as they relate to the previously indicated mediators of alliance, engagement, compliance and development of CBT skills.

As noted by Rutherford and Roose (2013), expectancy has largely been conceived as an unwanted factor to be controlled for in antidepressant research. Our results of robust negative associations between expectancy (both optimistic and pessimistic) at week 8 and post-treatment depression support a possible mechanistic role of this factor in pharmacotherapy treatment. Notably, previous studies on this topic are scant, but existing work points to similar mechanisms as those evidenced in CBT, specifically therapeutic alliance (Meyer et al., 2002). As suggested by Rutherford et al. (2017), future work is needed to better explicate the influence of expectancy in pharmacotherapy, potentially to inform revisions to existing clinical management protocols to incorporate optimization of expectancy effects (Rutherford et al., 2017).

4.3. Limitations

The results of this investigation meaningfully extend the literature on outcome expectancy; however, this study is not without limitations. First, our smaller sample size limited the complexity of cross-lagged models we were able to estimate. The nature of the data (i.e., different trajectories across treatments) dictated the use of group-specific analyses, which further limited our statistical power. Moreover, the pessimistic expectancy growth model in the pharmacotherapy group was uninterpretable, as was the optimistic expectancy model including the random slope variable. Inadmissible solutions may suggest misspecification due to failure to include relevant predictors in our models (Kline, 2011). Moreover, previous simulation studies have shown that a number of factors, including small sample size and fewer assessments, may increase the likelihood of inadmissible solutions when estimating growth (Diallo et al., 2014). Taken together, while the current results provide an important initial exploration of differential changes in outcome expectancy across different treatment modalities, future studies with more frequent assessments of outcome expectancy and larger sample sizes allowing for more complex modeling are needed. Second, our investigation of the role of outcome expectancy as a putative mechanism was circumscribed to exploring the cross-lagged associations in point-estimates of expectancy and depression during treatment, and we did not explore the specific role of *changes* in this construct. Future studies with larger sample sizes will be necessary to substantiate outcome expectancy as a mechanism by measuring the effect of change throughout treatment, particularly by capturing more frequent assessments early on. Moreover, as the parent randomized trial largely focused on the mediational role of cognition in therapeutic response, variables of alliance, treatment adherence (i.e., homework compliance and CBT skill acquisition for CBT, and pill counts and blood levels for pharmacotherapy) – possible mediators of the expectancy-outcome link – were not able to be included. As such, we were unable to explore the putative associations of these proposed processes. Nevertheless, our use of a randomized design in this study enabled exploration of treatment-specific roles of outcome expectancy in the two most widely used treatments for MDD.

5. Conclusions

To the best of our knowledge, this is the first study to explore changes in outcome expectancy throughout the course of antidepressant treatment, as well as to compare the relation between expectancy and outcome across CBT and pharmacotherapy. The results of this study reiterate the critical role of pre-treatment expectancy for CBT response, and support a possible mechanistic role in antidepressant response. Indeed, the status of outcome expectation as a common factor in psychotherapy may be extended to represent a possible common mechanism in the treatment of depression overall. However, larger studies with more frequent assessments of during treatment expectancy will be needed to validate these results. Clinically, these results implicate outcome expectancy as a potentially important target for both psychological and pharmacological treatments for MDD. More specifically, as patients presenting with higher optimistic beliefs before treatment begins benefit from CBT, efforts to bolster optimism will be crucial early in treatment. Initial sessions (e.g., educating the patient to the model, goal setting) might include traditional psychoeducation regarding research evidence for the benefits of CBT as well as a strength-based approach with a focus on patient resilience, to target optimistic outcome expectancies even further. For pharmacotherapy, clinical management protocols may benefit from strategies to enhance optimism applied throughout the course of treatment. Psychoeducation regarding the time course of medication effects and the opportunities to switch or augment pharmacotherapy as needed to maximize therapeutic benefit, and working with patients to increase compliance, motivation, and ultimately, their own sense of responsibility and empowerment in their recovery, may be useful. Moreover, an important future direction includes greater attention to expectancy type (optimistic versus pessimistic) in studies aiming to further explicate this construct.

Conflicts of interest

All authors report no conflicts of interest.

Contributors

LCQ was the primary investigator of the original trial and oversaw study design. Authors DJAD, RMB, DSSL, and LNR assisted in the study design and data collection in the original trial. TT and LCQ were involved in generating the research questions in the current study, as well as conducting analyses, interpreting results and drafting the initial manuscript. All authors contributed to and approved the final manuscript.

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